09/777,732

WEST

Freeform Search

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Term: 114 and transplant$5

Display: 10 **Documents in Display Format:** - **Starting with Number** 1

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Search History

DATE: Friday, February 21, 2003 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L16	l14 and transplant$5	3	L16
L15	L14 and transplat$5	0	L15
L14	L13 and cytoprotect$3	8	L14
L13	l6 and gene expres$4	368	L13
L12	L11 and rejection$1	1	L12
L11	L10 and (evaluat$4 or diagnos$3)	4	L11
L10	l8 and transplant$5	4	L10
L9	l7 and cytoprotective	1	L9
L8	l6 and cytoprotective	13	L8
L7	L6 and (evaluat$$ near5 acute near5 transplant$4 near5 rejection$1)	1	L7
L6	heme oxygenase or A20 or BcL-X	3269	L6
L5	L4 and (heme oxygenase or A20)	0	L5
L4	L3 and (gene near5 expres$4)	1	L4
L3	L2 and (transplant$4 near5 rejection)	1	L3
L2	cytoprotective gene$1	2	L2
L1	cytoprotective gene41	0	L1

END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 4 of 4 returned.

☐ 1. 6140484 . 10 Mar 98; 31 Oct 00. Bax .omega. protein and methods. Bitler; Catherine Mastroni, et al. 536/23.1; 435/320.1. C07H021/02.

☐ 2. 5804551 . 12 Nov 96; 08 Sep 98. Pretraumatic use of hemoglobin. Burhop; Kenneth E.. 514/6; 424/529 424/530 530/385 530/829. A61K037/02 A61K035/14.

☐ 3. 5770690 . 15 Mar 96; 23 Jun 98. Bax omega protein and methods. Bitler; Catherine Mastroni, et al. 530/324; 530/329 530/350. C07K014/00 C07K007/00.

☐ 4. WO 200181916 A2 AU 200157161 A US 20020132235 A1 . Evaluating acute transplant rejection in a host especially in a recipient of a urinary system graft, by determining a heightened magnitude of expression of genes in rejection-associated gene clusters. AVIHINGSANON, Y, et al. C12Q001/68 G01N033/48.

Generate Collection | Print

Term	Documents
EVALUAT$4	0
EVALUAT.DWPI,EPAB,JPAB,USPT.	29
EVALUATABLE.DWPI,EPAB,JPAB,USPT.	407
EVALUATABLY.DWPI,EPAB,JPAB,USPT.	1
EVALUATAD.DWPI,EPAB,JPAB,USPT.	2
EVALUATAE.DWPI,EPAB,JPAB,USPT.	1
EVALUATAED.DWPI,EPAB,JPAB,USPT.	2
EVALUATAION.DWPI,EPAB,JPAB,USPT.	2
EVALUATD.DWPI,EPAB,JPAB,USPT.	10
EVALUATE.DWPI,EPAB,JPAB,USPT.	116649
EVALUATEBUF.DWPI,EPAB,JPAB,USPT.	2
(L10 AND (EVALUAT$4 OR DIAGNOS$3)).USPT,JPAB,EPAB,DWPI.	4

There are more results than shown above. Click here to view the entire set.

Previous Page Next Page

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FILE 'HOME' ENTERED AT 16:16:36 ON 21 FEB 2003

=> file caplus medline biosis
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.63       0.63

FILE 'CAPLUS' ENTERED AT 16:18:36 ON 21 FEB 2003
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FILE 'MEDLINE' ENTERED AT 16:18:36 ON 21 FEB 2003

FILE 'BIOSIS' ENTERED AT 16:18:36 ON 21 FEB 2003
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=> s cytoprot5 (10a) gene (10a) expres4
4 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
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"HELP COMMANDS" at an arrow prompt (=>).

=> s cytoprot#####(10a)gene(10a)expres####
L1            9 CYTOPROT#####(10A) GENE(10A) EXPRES####

=> s l1 and transplant#####
L2            0 L1 AND TRANSPLANT#####

=> s cytoprot#####(10a)transplant#####
L3            0 CYTOPROT#####(10A) TRANSPLANT#####

=> s cytoprot##### and transplant#####
L4           11 CYTOPROT##### AND TRANSPLANT#####

=> s l4 and gene and expres#####
L5            0 L4 AND GENE AND EXPRES#####

=> dup rem l4
PROCESSING COMPLETED FOR L4
L6             7 DUP REM L4 (4 DUPLICATES REMOVED)

=> d l6 bib ab 1-7

L6   ANSWER 1 OF 7     MEDLINE                              DUPLICATE 1
AN   2002677669     IN-PROCESS
DN   22325568   PubMed ID: 12439700
TI   Amifostine reduces mucosal damage after high-dose melphalan conditioning
     and autologous peripheral blood progenitor cell transplantation
     for patients with multiple myeloma.
AU   Thieblemont C; Dumontet C; Saad H; Roch N; Bouafia F; Arnaud P; Hequet O;
     Espinouse D; Salles G; Roy P; Eljaafari-Corbin A; Du Manoir-Baumgarten C;
     Coiffier B
CS   Haematology Department, Centre Hospitalier Lyon-Sud, Pierre-Benite,
     France.
SO   BONE MARROW TRANSPLANTATION, (2002 Dec) 30 (11) 769-75.
     Journal code: 8702459. ISSN: 0268-3369.
CY   England: United Kingdom
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   IN-PROCESS; NONINDEXED; Priority Journals
ED   Entered STN: 20021120
```

Last Updated on STN: 20021213

AB High-dose melphalan (HDM) has been adopted as standard therapy in the treatment of multiple myeloma. This treatment is associated with non-selective cytotoxicity, causing oral mucositis as the major non-hematological side-effect. Amifostine is a **cytoprotector** which prevents toxicity induced by anticancer therapy. We prospectively compared two groups of patients who either received (group A, n = 21) or did not receive (group B, n = 20) amifostine (740 mg/m(2)) before HDM (200 mg/m(2)) followed by autologous peripheral blood progenitor cell **transplantation**. The occurrence of severe oral mucositis was significantly decreased in group A in comparison to group B (33% vs 65%, P < 0.05). Six patients in group A required opioid analgesic therapy during a mean period of 4.8 days as compared to eight patients for 6.5 days in group B (P = NS). Delayed vomiting was less frequent in group A (43% vs 70%, P = 0.07) and significantly less severe in group A (grade 2-4) vomiting: two patients vs nine patients, P < 0.02). No difference was observed between the two groups in either hematological toxicity after HDM or in response rate. Grade I emesis was the only immediate side-effect observed after amifostine administration. We conclude that amifostine can reduce mucositis induced by HDM.

L6 ANSWER 2 OF 7 MEDLINE DUPLICATE 2
AN 2001444331 MEDLINE
DN 21383027 PubMed ID: 11490368
TI A tripartite anoikis-like mechanism causes early isolated islet apoptosis.
AU Thomas F; Wu J; Contreras J L; Smyth C; Bilbao G; He J; Thomas J
CS Division of Transplantation, Department of Surgery, University of Alabama Medical Center, Birmingham, AL 35294-0012, USA.
NC U19-DK7958 (NIDDK)
SO SURGERY, (2001 Aug) 130 (2) 333-8.
Journal code: 0417347. ISSN: 0039-6060.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200109
ED Entered STN: 20010813
Last Updated on STN: 20010910
Entered Medline: 20010906

AB BACKGROUND: This study examines the mechanisms of early isolated islet apoptosis (II-APO) and loss of functional islet mass. METHODS: Rhesus islets were isolated for **transplantation**, and an aliquot was used for in vitro molecular studies of II-APO. These studies used Western blotting to examine caspase activation and perinuclear envelope protein cleavage that are associated with II-APO and used immunofluorescence analysis of Annexin V and mitochondrial permeability index to examine spontaneous and tripartite anoikis-like (TRAIL) mechanism--induced II-APO. RESULTS: Caspase 6 was prominently activated in association with spontaneous II-APO, which occurred after overnight culture. In contrast, caspase 7, 8, and 9 were not activated. Cleavage of focal adhesion kinase and Lamin, substrates of caspase 6, was also evident in spontaneous II-APO. II-APO was exaggerated by the addition of the TRAIL mechanism. The TRAIL mechanism--induced II-APO was blocked by the caspase 6 inhibitor, VEID, and by the soluble fusion proteins, DR4 or DR5, which act as decoy receptors. In vivo studies in diabetic severe combined immunodeficiency disease mice showed that rhesus islets were **cytoprotected** by either ex vivo gene transfer of Bcl-2 or treatment of the isolated islet with VEID. CONCLUSIONS: These studies suggest 3 major mechanisms involved in II-APO: caspase 6 activation, a TRAIL-induced apoptosis pathway, and the mitochondrial-associated apoptosis pathway. Inhibition of these II-APO pathways may improve isolated islet survival and reduce functional islet mass loss, which compromises the stable reversal of diabetes.

L6 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 3

AN 2000:645347 CAPLUS
DN 134:125639
TI Amifostine can reduce mucosal damage after high-dose melphalan conditioning for peripheral blood progenitor cell autotransplant: a retrospective study
AU Capelli, D.; Santini, G.; De Souza, C.; Poloni, A.; Marino, G.; Montanari, M.; Lucesole, M.; Brunori, M.; Massidda, D.; Offidani, M.; Leoni, P.; Olivieri, A.
CS Department of Haematology, University of Ancona, Ancona, 60020, Italy
SO British Journal of Haematology (2000), 110(2), 300-307
CODEN: BJHEAL; ISSN: 0007-1048
PB Blackwell Science Ltd.
DT Journal
LA English
AB Amifostine (WR-2721: Ethyol) is a well-known **cytoprotector**, but a possible role in preventing extra-haematol. toxicity after high-dose therapy (HDT) has never been investigated. We compared two historical groups of patients who either received (group A, n = 35) or did not receive (group B, n = 33) amifostine (740 mg/m2) before high-dose (HD) melphalan, followed by autologous infusion of peripheral blood progenitor cells (PBPCs). Amifostine was well tolerated at this dose level. Emesis grade 1-2 was the most important side-effect, but the interruption of infusion was never required. The incidence and median duration of severe mucositis (grade 3-4) was 21% and 0 d (range 0-11 d) in group A and 53% and 7 d (range 0-11 d) in group B. The duration of analgesic therapy was also significantly lower in group A (0 d; range 0-12) than in group B (6 d, range 0-20) (P = 0.0001). Severe diarrhea (3% vs. 25%; P = 0.01) and emesis (9% vs. 34%; P = 0.01) were also reduced in group A in comparison with group B. No differences were obsd. between the two groups for haematol. recovery. This retrospective study strongly suggests that amifostine can reduce severe mucositis and the use of analgesic drugs in this setting. A randomized study is warranted to confirm these preliminary results.
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 7 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1999:155422 BIOSIS
DN PREV199900155422
TI Prevention of endothelial cell activation in hamster-to-rat cardiac xenografts by gene transfection of BCL-2.
AU Kobayashi, Y.; Fukushima, N.; Ohtake, S.; Sawa, Y.; Nishimura, M.; Sakaguchi, T.; Miyagawa, S.; Matsuda, H.
CS Osaka Univ., Suita, Osaka Japan
SO Journal of Heart and Lung Transplantation, (Jan., 1999) Vol. 18, No. 1, pp. 66.
Meeting Info.: Nineteenth Annual Meeting and Scientific Sessions of the International Society for Heart and Lung Transplantation San Francisco, California, USA April 21-24, 1999 International Society for Heart and Lung Transplantation
. ISSN: 1053-2498.
DT Conference
LA English

L6 ANSWER 5 OF 7 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1996:396218 BIOSIS
DN PREV199699118574
TI L-arginine can attenuate warm ischemic injury in the rat kidney and nitric oxide production in the preserved kidney.
AU Kin, S. (1); Sasaki, T.; Gu, K.; Saitoh, Y.; Nagami, H.; Iwasaki, S.; Nakayama, K.; Tamura, K.
CS (1) First Dep. Surg.,, Shimane Med. Univ.,, 89-1 Enyacho, Izumo, Shimane 693 Japan
SO Transplantation Proceedings, (1996) Vol. 28, No. 3, pp. 1889-1890.

Meeting Info.: Fourth International Congress of the Asian Transplantation Society Seoul, Korea August 27-30, 1995
ISSN: 0041-1345.
DT Conference
LA English

L6 ANSWER 6 OF 7 MEDLINE DUPLICATE 4
AN 95162968 MEDLINE
DN 95162968 PubMed ID: 7859173
TI Purpurogallin as a **cytoprotector** of cultured rabbit corneal endothelium.
AU Rootman D S; Bindish R; Zeng L H; Hasany S M; Wu T W
CS Department of Ophthalmology, University of Toronto, Ont.
SO CANADIAN JOURNAL OF OPHTHALMOLOGY, (1994 Oct) 29 (5) 220-3.
Journal code: 0045312. ISSN: 0008-4182.
CY Canada
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199503
ED Entered STN: 19950404
Last Updated on STN: 19950404
Entered Medline: 19950320
AB We examined the protective properties of purpurogallin, a naturally occurring phenol, in delaying necrosis of cultured corneal endothelial cells caused by oxygen free radicals. Endothelial cell cultures were prepared from New Zealand white rabbits using microcarrier cell culture techniques. Corneal endothelial cells were treated with hypoxanthine (2 mM) and xanthine oxidase (67 IU/L) to generate free radicals. The criteria for cell necrosis were cytoplasmic shrinkage, dissolution of plasma membranes and presence of "haloes" around the cells on phase contrast microscopy, confirmed by transmission electron microscopy. More than 95% of second-generation cells exhibited morphologic evidence of necrosis within 4.62 +/- 0.82 minutes after exposure to oxyradicals. The addition of purpurogallin (0.25 or 1.0 mM) significantly increased time to cell necrosis to 8.18 +/- 0.83 and 11.59 +/- 1.71 minutes respectively ($p < 0.05$). Further studies are under way to determine whether purpurogallin may be useful in preventing endothelial cell damage in corneas preserved for corneal **transplantation**.

L6 ANSWER 7 OF 7 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1983:254514 BIOSIS
DN BA76:12006
TI HORMONALLY RESPONSIVE VS. UNRESPONSIVE PROGRESSION OF PROSTATIC CANCER TO ANTI ANDROGEN THERAPY AS STUDIED WITH THE DUNNING R-3327-AT AND R-3327-G RAT ADENO CARCINOMAS.
AU ISAACS J T
CS JOHNS HOPKINS ONCOL. CENT., JOHNS HOPKINS UNIV., BALTIMORE, MD. 21205.
SO CANCER RES, (1982) 42 (12), 5010-5014.
CODEN: CNREA8. ISSN: 0008-5472.
FS BA; OLD
LA English
AB The present study has compared the response to antiandrogen therapy of the serially **transplantable** Dunning R-3327-AT (AT) vs. Dunning R-3327-G (G) rat prostatic adenocarcinoma. Castration or chemical antiandrogen therapy (i.e., **cytoproterone** acetate and diethylstilbestrol) of rats bearing established AT or G tumors results in neither regression of tumor volume nor a cessation of the continuous growth of either tumor. By these criteria, both the AT and G tumors progress following antiandrogen therapy. For the AT tumor, this progression is completely unresponsive to hormonal therapy, and thus such therapy does not increase survival of AT tumor-bearing rats. The AT tumor is therefore an example of hormonally unresponsive progression. In direct contrast, while the G tumor likewise progresses following antiandrogen

therapy, this therapy does induce a 1.8-fold decrease in the subsequent growth rate of the G tumor. This positive response during progression of the G tumor results in a 78% increase in the survival of G tumor-bearing rats treated with antiandrogen therapy. The G tumor is therefore an example of hormonally responsive progression. These results indicate neither that prostatic cancers which do not regress or cease growing following antiandrogen therapy can necessarily be considered hormonally unresponsive nor that antiandrogen of such tumors has been completely ineffective, since, as shown in the present study, such progression can be of either a hormonally unresponsive or responsive type. Regardless of which type of progression occurs, however, additional therapy is required to further increase survival. Such additional therapy should probably include the subsequent use of pharmacological doses of exogenous androgen, since, depending on the type of progression, such treatments can actually decrease survival.

```
=> d l6 4 5 kwic

L6   ANSWER 4 OF 7  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
IT   . . .
Organisms
        coronary artery: circulatory system, perfusion; endothelial cell:
        activation; heart: circulatory system
IT   Chemicals & Biochemicals
        xenoantibody: apoptosis; Bcl-2 gene: cytoprotector gene,
        transfection
IT   Methods & Equipment
        heart transplantation [Htx]: transplantation
        method; immunohistochemistry: histochemical method
IT   Miscellaneous Descriptors
        Meeting Abstract

L6   ANSWER 5 OF 7  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
IT   Miscellaneous Descriptors
        CHEMICAL COORDINATION AND HOMEOSTASIS/URINARY SYSTEM;
        CYTOPROTECTOR; KIDNEY PRESERVATION; L-ARGININE; MEETING PAPER;
        NITRIC OXIDE PRODUCTION; THERAPEUTIC METHOD; TRANSPLANTATION;
        TRANSPORT AND CIRCULATION/CARDIOVASCULAR SYSTEM; UROLOGIC DISEASE;
        VASCULAR DISEASE; WARM ISCHEMIC INJURY


=> s heme oxygenase or A20 or BcL
L7       55791 HEME OXYGENASE OR A20 OR BCL

=> s l7 and transplat#####
L8           8 L7 AND TRANSPLAT#####

=> s l8 and gene expres####
   1 FILES SEARCHED...
L9           3 L8 AND GENE EXPRES####

=>

=> d l9 1-3 bib ab kwic

L9   ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2003 ACS
AN   2002:465747  CAPLUS
DN   137:41724
TI   CDDO (2-cyano-3,12-dioxoolean-1,9-dien-28-oic acid) compounds and
     combinations with other chemotherapeutics for the treatment of cancer and
     graft vs. host disease
IN   Konopleva, Marina; Andreef, Michael; Sporn, Michael
PA   Board of Regents of the University of Texas System, USA
```

SO PCT Int. Appl., 184 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002047611	A2	20020620	WO 2001-US44541	20011128
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2002043246	A5	20020624	AU 2002-43246	20011128
PRAI	US 2000-253673P	P	20001128		
	WO 2001-US44541	W	20011128		

AB CDDO compds. in combination with other chemotherapeutic agents induce and potentiate cytotoxicity and apoptosis in cancer cells. One class of chemotherapeutic agents include retinoids. Cancer therapies based on these combination therapies are provided. Also provided are methods to treat graft vs. host diseases using the CDDO compds.

IT Nucleic acids
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**Bcl**-2-encoding; CDDO compds. and combinations with other chemotherapeutics for treatment of cancer and graft vs. host disease)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**Bcl**-2; CDDO compds. and combinations with other chemotherapeutics for treatment of cancer and graft vs. host disease)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**Bcl**-xL; CDDO compds. and combinations with other chemotherapeutics for treatment of cancer and graft vs. host disease)

IT **Gene**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**expression**; CDDO compds. and combinations with other chemotherapeutics for treatment of cancer and graft vs. host disease)

IT 50-18-0, Cyclophosphamide 50-76-0, Dactinomycin 51-21-8, 5-Fluorouracil 51-75-2, Mechlorethamine 52-24-4, Thiotepa 55-98-1, Busulfan 57-22-7, Vincristine 59-05-2, Methotrexate 114-70-5, Sodium phenylacetate 147-94-4, Ara-C 148-82-3, Melphalan 154-93-8, Carmustine 156-54-7, Sodium butyrate 302-79-4, all-trans-Retinoic acid 305-03-3, Chlorambucil 645-05-6, Hexamethylmelamine 671-16-9, Procarbazine 865-21-4, Vinblastine 1404-00-8, Mitomycin 2353-33-5, Decitabine 3778-73-2, Ifosfamide 4342-03-4, Dacarbazine 5300-03-8, 9-cis-Retinoic acid 7689-03-4, Camptothecin 7722-84-1, Hydrogen peroxide, biological studies 10540-29-1, Tamoxifen 11056-06-7, Bleomycin 13010-20-3, Nitrosurea 13010-47-4, Lomustine 13909-09-6, Semustine 14913-33-8, **Transplatin** 15663-27-1, Cisplatin 18378-89-7, Plicamycin 18883-66-4, Streptozocin 20830-81-3, Daunorubicin 23214-92-8, Doxorubicin 25316-40-9, Adriamycin 29767-20-2, Teniposide 33069-62-4, Taxol 33419-42-0, Etoposide 41575-94-4, Carboplatin 65271-80-9, Mitoxantrone 65646-68-6, Fenretinide 92689-49-1, Annamycin 100629-51-4, Bryostatin 104987-11-3, Tacrolimus 110417-88-4, Dolastatin 10 125316-60-1, CD437 153559-49-0, LGD1069 153559-76-3, LG100268 218600-44-3D, derivs. 220578-59-6, Mylotarg
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(CDDO compds. and combinations with other chemotherapeutics for treatment of cancer and graft vs. host disease)

L9 ANSWER 2 OF 3 MEDLINE
AN 2001522656 MEDLINE
DN 21454022 PubMed ID: 11568363
TI Assessment of cisplatin-induced nephrotoxicity by microarray technology.
AU Huang Q; Dunn R T 2nd; Jayadev S; DiSorbo O; Pack F D; Farr S B; Stoll R E; Blanchard K T
CS Department of Toxicology, Boehringer Ingelheim Pharmaceuticals, Inc., 900 Ridgebury Road, P.O. Box 368, Ridgefield, Connecticut 06877-0368, USA.. qhuang@rdg.boeringer-ingelheim.com
SO TOXICOLOGICAL SCIENCES, (2001 Oct) 63 (2) 196-207.
Journal code: 9805461. ISSN: 1096-6080.
CY United States
DT (EVALUATION STUDIES)
Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200112
ED Entered STN: 20010925
Last Updated on STN: 20020122
Entered Medline: 20011212
AB Microarrays are a new technology used to study global **gene expression** and to decipher biological pathways. In the current study, microarrays were used to examine **gene expression** patterns associated with cisplatin-mediated nephrotoxicity. Sprague-Dawley rats received either single or seven daily ip doses of cisplatin (0.5 or 1 mg/kg/day) or the inactive isomer **transplatin** (1 or 3 mg/kg/day). Histopathological evaluation revealed renal proximal tubular necrosis in animals that received cisplatin for 7 days, but no hepatotoxic findings. Microarray analyses were performed using rat specific arrays containing 250 toxicity-related genes. Prominent **gene expression** changes were observed only in the kidneys of rats that received cisplatin for 7 days. Mechanistically, the **gene expression** pattern elicited by cisplatin (e.g., Bax upward arrow and SMP-30 downward arrow) suggested the occurrence of apoptosis and the perturbation of intracellular calcium homeostasis. The induction of multidrug resistance genes (MDR1 upward arrow, P-gp upward arrow) and tissue remodeling proteins (clusterin upward arrow, IGFBP-1 upward arrow, and TIMP-1 upward arrow) indicated the development of cisplatin resistance and tissue regeneration. Select **gene expression** changes were further confirmed by TaqMan analyses. **Gene expression** changes were not observed in the liver following cisplatin administration. In contrast to these in vivo findings, studies using NRK-52E kidney epithelial cells and clone-9 liver cells suggested that liver cells were more sensitive to cisplatin treatment. The discrepancies between the in vivo and in vitro results suggest that caution should be taken when extrapolating data from in vivo to in vitro systems. Nonetheless, the current study elucidates the biochemical pathways involved in cisplatin toxicity and demonstrates the utility of microarrays in toxicological studies.
AB Microarrays are a new technology used to study global **gene expression** and to decipher biological pathways. In the current study, microarrays were used to examine **gene expression** patterns associated with cisplatin-mediated nephrotoxicity. Sprague-Dawley rats received either single or seven daily ip doses of cisplatin (0.5 or 1 mg/kg/day) or the inactive isomer **transplatin** (1 or 3 mg/kg/day). Histopathological evaluation revealed renal proximal tubular necrosis in animals that received cisplatin for 7 days, but no hepatotoxic findings. Microarray analyses were performed using rat specific arrays containing 250 toxicity-related genes. Prominent **gene expression** changes were observed only in the kidneys of rats that received cisplatin for 7 days. Mechanistically, the **gene expression** pattern elicited by cisplatin (e.g., Bax upward arrow and SMP-30 downward arrow) suggested the occurrence of apoptosis and the

perturbation. . . (clusterin upward arrow, IGFBP-1 upward arrow, and TIMP-1 upward arrow) indicated the development of cisplatin resistance and tissue regeneration. Select **gene expression** changes were further confirmed by TaqMan analyses. **Gene expression** changes were not observed in the liver following cisplatin administration. In contrast to these in vivo findings, studies using NRK-52E. . .

CT . . .

metabolism

Cell Line
Cisplatin: AD, administration & dosage
*Cisplatin: TO, toxicity
Epithelial Cells: DE, drug effects
Epithelial Cells: ME, metabolism
***Gene Expression: DE, drug effects**
Genes, MDR: DE, drug effects
Glycoproteins: ME, metabolism
Hepatocytes: DE, drug effects
Injections, Intraperitoneal
Insulin-Like Growth-Factor. . . metabolism
Liver: DE, drug effects
Molecular Chaperones: ME, metabolism
*Oligonucleotide Array Sequence Analysis
Polymerase Chain Reaction
Proto-Oncogene Proteins: ME, metabolism
Proto-Oncogene Proteins c-bcl-2: ME, metabolism
Random Allocation
Rats
Rats, Sprague-Dawley
Stereoisomerism
Time Factors
Tissue-Inhibitor of Metalloproteinase-1: ME, metabolism

RN **14913-33-8 (transplatin)**; 15663-27-1 (Cisplatin)

CN. . . 0 (Calcium-Binding Proteins); 0 (Glycoproteins); 0 (Insulin-Like Growth-Factor Binding Protein 1); 0 (Molecular Chaperones); 0 (Proto-Oncogene Proteins); 0 (Proto-Oncogene Proteins c-**bcl**-2); 0 (Tissue-Inhibitor of Metalloproteinase-1); 0 (clusterin); 0 (regucalcin)

L9 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

AN 2000:294831 BIOSIS

DN PREV200000294831

TI Effect of harmonious static magnet field (HSMF) on expression of apoptosisralated gene **Bcl**-2 and Bax protein in Walker-256-implanted rats.

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SO Xi'an Yike Daxue Xuebao, (Apr., 2000) Vol. 21, No. 2, pp. 100-103. print. ISSN: 0258-0659.

DT Article

LA Chinese

SL Chinese; English

AB Objective: To study the expression of **Bcl**-2 and Bax proteins, to explore the molecular mechanism of antitumor effect and supply data and vidences in the treatment of tumors on Walker-256-implanted rats following HSMF. Methods: Walker-256 carcinoma cells line was **transplated** into the right thighs of 460 SD rats, 400 of 460 samples were divided into 2 qroups randomly: the tumor experiment group and the tumor control group on the basis of completely random principle after being fed with 2 weeks on routine. The experiment group were exposed to differential power and time magetic field every day, to continue 2 weeks and observe the survival rate of rats, then to measure volume and wet weighty of the body of sarcoma, in the last HE and ABC immunohistochemistry staining method were used. Results: In the period of experiment, it was found that SD rats

survival rate (84.28%) was increased, sarcoma volume and wet weighty were significantly decreased when compared with the tumor control group (P<0. 05). The pathology examination showed that the number of apoptosis were significantly increased. The positive expression rate (18%) of **Bcl** -2 protein were decreased, the expression of Bax gene was contrary to **Bcl**-2 gene and the ratio of **Bcl**-2/Bax was significantly decreased. There was significant differenced in 2 groups (P<0. 05). Conclusion: HSM can decrease the expression of **Bcl**-2 gene, increase the expression of Bax and decrease the ratio of **Bcl** -2/Bax. It suggested that it achieved its antitumor effect by inducing apoptosis of SD rats **transplation** sarcoma cells. HSM showed a potentiation of the antitumor effect as a clinically new useful tool.

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IT . . .
(Biochemistry and Molecular Biophysics); Radiation Biology; Tumor Biology

IT Diseases
sarcoma: neoplastic disease

IT Chemicals & Biochemicals
Bax protein: expression; **Bcl**-2 protein: expression; rat bax gene (Muridae): apoptosis-related **gene**, **expression**; rat **bcl**-2 gene (Muridae): apoptosis-related **gene**, **expression**

IT Alternate Indexing
Sarcoma (MeSH)

```
=> s l7 and (allograft# or xenograft#)
L10         597 L7 AND (ALLOGRAFT# OR XENOGRAFT#)

=> s l10 and gene expres####
   1 FILES SEARCHED...
L11          73 L10 AND GENE EXPRES####

=> s l1 and cytoprotective
L12           1 L1 AND CYTOPROTECTIVE

=> d l12 bib ab kwic

L12  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2003 ACS
AN   2002:339284  CAPLUS
DN   138:33258
TI   Activation of the nuclear transcription factor .kappa.B (NF.kappa.B) and
     differential gene expression in U87 glioma cells after
     exposure to the cytoprotector amifostine
AU   Kataoka, Yasushi; Murley, Jeffrey S.; Khodarev, Nikolai N.; Weichselbaum,
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Ralph R.; Grdina, David J.

CS Department of Radiation and Cellular Oncology, University of Chicago, Chicago, IL, USA

SO International Journal of Radiation Oncology, Biology, Physics (2002), 53(1), 180-189
CODEN: IOBPD3; ISSN: 0360-3016
PB Elsevier Science Inc.
DT Journal
LA English

AB Purpose: Amifostine has been approved as a therapy to decrease the incidence of moderate-to-severe xerostomia in patients undergoing postoperative radiation treatment for head-and-neck cancer. As a reducing agent capable of participating in intracellular reductive/oxidative processes, it has the potential to affect redox-sensitive transcription factors and gene expression. Amifostine's active free thiol WR-1065 was investigated to det. its effect on nuclear transcription factor .kappa.B (NF.kappa.B) activation and subsequent gene expression in U87 glioma cells. Methods and Materials: The human glioma cell line U87 was grown to confluency and then exposed to WR-1065 at a concn. of 40 .mu.M for times ranging from 30 min to 24 h. Changes in cell cycle were monitored by flow cytometry. The effect of WR-1065 on NF.kappa.B activation was detd. by a gel shift assay. Changes in gene expression as a function of time of exposure to WR-1065 were detd. by Northern blot and the Atlas Human cDNA Expression Array (Clontech, Palo Alto, CA). Changes in gene expression using the Atlas Array were verified by reverse transcriptase-polymerase chain reaction (RT-PCR) with gene-specific primers. Results: Exposure of U87 cells to 40 .mu.M WR-1065 resulted in a marked activation of NF.kappa.B between 30 min and 1 h after treatment. Expression of MnSOD, an NF.kappa.B-responsive gene, was enhanced by over 2-fold after 16 h of treatment and remained elevated at 24 h. During this period of time, no changes in cell cycle distribution were obsd. To assess changes in the expression levels of NF.kappa.B-responsive genes as a function of WR-1065 exposure, cDNA arrays contg. 49 genes identified as having DNA-binding motifs for NF.kappa.B were used. Only five genes were found to be significantly affected at 1, 4, and/or 16 h of treatment. GST-3 and c-myc were repressed up to 2- and 4-fold, resp. The expression levels of IL-2Ra, RANTES, and c-myb, in contrast, were enhanced up to 14-, 3-, and 2-fold, resp. The remaining genes having NF.kappa.B-responsive elements in their promoter regions were either not expressed (20 genes) or were not affected (24 genes) by exposure to WR-1065. Conclusions: The redox-sensitive transcription factor NF.kappa.B can be activated in U87 glioma cells by the active thiol form of the cytoprotector amifostine. Activation of NF.kappa.B by the antioxidant WR-1065 is accompanied by a reduced expression of the oncogene c-myc and an enhanced expression of the antioxidant gene MnSOD, a gene whose expression in tumor cells is relatively low, but when overexpressed has been correlated with a suppression of the malignant phenotype. Activation of NF.kappa.B by WR-1065, however, results in selective rather than global changes in the expression of genes contg. NF.kappa.B-responsive elements.

RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Activation of the nuclear transcription factor .kappa.B (NF.kappa.B) and differential **gene expression** in U87 glioma cells after exposure to the **cytoprotector** amifostine

IT Antioxidants
Cell cycle
Cytoprotective agents
Gene expression profiles, animal
Human
(activation of nuclear transcription factor .kappa.B and differential gene expression in human U87 glioma cells after exposure to thiol form of cytoprotector amifostine)